VPP2 Rabbit pAb

CatalogNo: YN1516

Key Features

Host Species
- Rabbit

Reactivity
- Human,Mouse

Applications
- WB,ELISA

MW
- 94kD (Observed)

Isotype
- IgG

Recommended Dilution Ratios

WB 1:500-2000
ELISA 1:5000-20000

Storage

Storage*	-15°C to -25°C/1 year(Do not lower than -25°C)
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.

Basic Information

Clonality	Polyclonal

Immunogen Information

Immunogen	Synthesized peptide derived from part region of human protein
Specificity	VPP2 Polyclonal Antibody detects endogenous levels of protein.

Target Information

Gene name	ATP6V0A2

Protein Name V-type proton ATPase 116 kDa subunit a isoform 2 (V-ATPase 116 kDa isoform a2) (Lysosomal H(+)-transporting ATPase V0 subunit a2) (TJ6) (Vacuolar proton translocating ATPase 116 kDa subunit a isoform 2)

Organism	Gene ID	UniProt ID
Human	23545;	Q9Y487;
Mouse		P15920;

Cellular Localization Cell membrane; Multi-pass membrane protein. Endosome membrane. In kidney proximal tubules, also detected in subapical vesicles. .

Tissue specificity Astrocyte,Epithelium,Placenta,Prostate,

Function Caution:The N-terminus peptide may increase IL1B secretion by peripheral blood monocytes; however as this region is probably in the cytosol, the in vivo relevance of this observation needs to be confirmed.,Disease:Defects in ATP6V0A2 are a cause of wrinkly skin syndrome; (WSS) [MIM:278250]. WSS is rare autosomal recessive disorder characterized by wrinkling of the skin of the dorsum of the hands and feet, an increased number of palmar and plantar creases, wrinkled abdominal skin, multiple musculoskeletal abnormalities, microcephaly, growth failure and developmental delay.,Disease:Defects in ATP6V0A2 are the cause of cutis laxa type II (ARCL type II) [MIM:219200]. ARCL type II is an autosomal recessive disorder characterized by an excessive congenital skin wrinkling, a large fontanelle with delayed closure, a typical facial appearance with downslanting palpebral fissures, a general connective tissue weakness, and varying degrees of growth and developmental delay and neurological abnormalities. Some affected individuals develop seizures and mental deterioration later in life, whereas the skin phenotype tends to become milder with age. At the molecular level, this disorder belongs to the family of congenital disorders of glycosylation (CDG) and is characterized by the abnormal glycosylation of serum proteins.,Function:Part of the proton channel of V-ATPases. Essential component of the endosomal pH-sensing machinery. May play a role in maintaining the Golgi functions, such as glycosylation maturation, by controlling the Golgi pH.,PTM:Phosphorylated upon DNA damage, probably by ATM or ATR.,similarity:Belongs to the V-ATPase 116 kDa subunit family.,subcellular location:In kidney proximal tubules, also detected in subapical vesicles.,subunit:The V-ATPase is an heteromultimeric enzyme composed of at least thirteen different subunits. It has a membrane peripheral V1 sector for ATP hydrolysis and an integral V0 for proton translocation. The V1 sector comprises subunits A-H, whereas V0 includes subunits a, d, c, c', and c''. Directly interacts with PSCD2 through its N-terminal cytosolic tail in an intra-endosomal acidification-dependent manner. Disruption of this interaction results in the inhibition of endocytosis.,

Validation Data

Contact information

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